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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/913,731	10/19/2001	Philippe Amouyel	P07337US00	3795

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EXAMINER
SAKELARIS, SALLY A

ART UNIT	PAPER NUMBER
1634	

DATE MAILED: 09/22/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/913,731	AMOUYEL ET AL.	
	Examiner	Art Unit	
	Sally A Sakelaris	1634	

-- The MAILING DATE of this communicati n appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 October 2001* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-24* is/are pending in the application.

4a) Of the above claim(s) *1-12* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *13-24* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☒ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: .

DETAILED ACTION

Priority

Acknowledgement of this application as the national stage entry of PCT/EP00/01549, 2/17/2000 has been made. The filing date of the instant claims is deemed to be the filing date of the national stage entry of PCT/EP00/01549, 2/17/2000.

Claim Objections

A. Claims 15, 17, and 18 are objected to for the redundancy of the recitation of "sequence SEQ ID NO:". Applicant should note that "SEQ" stands for "sequence" and that appropriate correction is required.

B. Claim 18 is objected to over the recitation of "SUR1 gene combining nucleotide – 3 as showed in sequence SEQ ID NO: 1". It is not clear if applicant intended to claim SUR1 gene in combination with a single "-3 nucleotide", or if applicant intended to use the word "containing" instead of "combining". Appropriate correction is required.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

1. Claims 19 and 20 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

A. Claims 19 and 20 are indefinite over the recitation of "corresponding to said first fragment". The actual "corresponding" feature is not defined by the claim, the specification does not provide a standard for ascertaining the requisite degree and one of ordinary skill in the art would not be reasonably appraised of the scope of the invention. There is no fixed definition in the art for what constitutes a "corresponding" DNA sequence. It is unclear if the second fragment is corresponding to the first segment because the second fragment represents a sequence from the same patient but represents another allele at the same position, or alternatively if the second fragment corresponds to the first because they both comprise nucleotide –3, but are from different patients. It is unclear where the correspondence occurs, what it is, what effect the correspondence has or to what non-corresponding segments these segments are being compared. Applicant must amend the claims to clarify the exact way in which the first and second segments correspond to one another.

B. Claims 19 and 20 are further indefinite over the recitation of "said human sample." The phrase lacks antecedent basis as claim 13 recites a "sample from a NIDDM patient" but makes no prior reference to a human sample to which "said human sample" refers. It is therefore unclear to which sample claims 19 and 20 are referencing and appropriate correction is required.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

2. Claims 13, 14, 16, 19, 20, and 21are rejected under 35 U.S.C. 102(b) as being anticipated by Hansen et al.(Diabetes, April 1998).

With regard to claim 13, Hansen et al. teach that "a diminished acute insulin secretory response to intravenous glucose or sulfonylurea loads is a frequent feature of late-onset NIDDM"(Pg. 508). The reference further teaches this method comprising:

a) obtaining a sample from a NIDDM patient, said sample comprising nucleic acid molecules containing the fragment of the SUR1 gene comprising the nucleotide in position –3 of exon 16(Table 4, pg. 602),

b) detecting the presence or the absence of the –3t allele of exon 16, whereby the presence of at least one –3t allele identifies a NIDDM patient with a higher susceptibility toward sulfonylurea therapy(Entire document).

As a whole, the reference teaches that the "at risk genotype" for NIDDM is comprised by at least one –3t allele of exon 16, and that a characteristic of late onset NIDDM is a diminished response to sulfonylurea loads.

With regard to claim 14, Hansen et al. teach the above method further comprising prior to step b) the step of amplifying said nucleic acid molecules using amplification primers that selectively anneal to and amplify a portion of said gene comprising the nucleotide in position –3 of exon 16(Table 1 and Research Design and Methods section).

With regard to claim 16, Hansen et al. teach the above method wherein said detecting step b) comprises sequencing all or part of the sequence of intron 15 comprising said –3 nucleotide as "oligonucleotide sequences for PCR amplification of the 38 SUR1

exons and intron-exon boundaries were designed from the genomic SUR1 sequence"(Pg. 599).

With regard to claims 19, 20, and 21 Hansen et al. teach the above method wherein said detecting step b) comprises obtaining a first gene fragment comprising nucleotide –3 of exon 16 isolated from said human sample and a second gene fragment comprising nucleotide –3 of exon 16 isolated from said human sample and a second gene fragment comprising nucleotide –3c and –3t of exon 16, said second fragment corresponding to said first fragment, forming single-stranded DNA from said SUR1 gene fragment and from said second SUR1 gene fragment, electrophoresing said single-stranded DNAs on said gel to determine if said single-stranded DNA from said first SUR1 gene fragment is shifted relative to said second SUR1 gene fragment, and optionally sequencing said single-stranded DNA from said first SUR1 gene fragment having a shift in mobility.(See Research Design and Methods and specifically Pg. 599 and results on Page 601)

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

3. Claims 15, 17, and 18 are rejected under 35 U.S.C. 103(a) as being unpatentable over Hansen et al(Diabetes, April 1998) in view of Gonzalez et al(Genbank accession no. L78223; SUR1 gene, exon 17, June 1996) and further in view of Buck et al (Biotechniques (1999) 27(3):528-536).

With regard to claim 13, Hansen et al. teach that "a diminished acute insulin secretory response to intravenous glucose or sulfonylurea loads is a frequent feature of late-onset NIDDM"(Pg. 508). The reference further teaches this method comprising:

a) obtaining a sample from a NIDDM patient, said sample comprising nucleic acid molecules containing the fragment of the SUR1 gene comprising the nucleotide in position –3 of exon 16(Table 4, pg. 602),

b) detecting the presence or the absence of the –3t allele of exon 16, whereby the presence of at least one –3t allele identifies a NIDDM patient with a higher susceptibility toward sulfonylurea therapy(Entire document).

As a whole, the reference teaches that the "at risk genotype" for NIDDM is comprised by at least one –3t allele of exon 16, and that a characteristic of late onset NIDDM is a diminished response to sulfonylurea loads.

With regard to claim 14, Hansen et al. teach the above method further comprising prior to step b) the step of amplifying said nucleic acid molecules using amplification primers that selectively anneal to and amplify a portion of said gene comprising the nucleotide in position –3 of exon 16(Table 1 and Research Design and Methods section).

With regard to claim 16, Hansen et al. teach the above method wherein said detecting step b) comprises sequencing all or part of the sequence of intron 15 comprising said –3 nucleotide as "oligonucleotide sequences for PCR amplification of the 38 SUR1 exons and intron-exon boundaries were designed from the genomic SUR1 sequence"(Pg. 599).

With regard to claims 19, 20, and 21 Hansen et al. teach the above method wherein said detecting step b) comprises obtaining a first gene fragment comprising nucleotide –3 of exon 16 isolated from said human sample and a second gene fragment comprising nucleotide –3 of exon 16 isolated from said human sample and a second gene fragment comprising nucleotide –3c and –3t of exon 16, said second fragment corresponding to said first fragment, forming single-stranded DNA from said SUR1 gene fragment and from said second SUR1 gene fragment, electrophoresing said single-stranded DNAs on said gel to determine if said single-stranded DNA from said first SUR1 gene fragment is shifted relative to said second SUR1 gene fragment, and optionally sequencing said single-stranded DNA from said first SUR1 gene fragment

having a shift in mobility.(See Research Design and Methods and specifically Pg. 599 and results on Page 601)

With regard to claims 15, 17, and 18, Hansen teaches amplification primers specific to exon 16 and the surrounding intron/exon boundaries in addition to teaching a method comprising performing a restriction endonuclease digestion for genotype confirmation(Pg. 601).

Hansen et al. does not teach a probe that selectively hybridizes to a portion of intron 15 of SUR1 gene nor does the Hansen et al. reference teach one of the particular oligonucleotides of SEQ ID Nos: 1-3, but Hansen does teach that primer selection "for PCR amplification of the 38 SUR1 exons and intron-exon boundaries were designed from the genomic SUR1 sequence (G.Gonzalez, Genbank accession no L78208-L78223) to generate PCR products"(Pg. 599).

Gonzalez teaches the SUR1 gene's DNA sequence for SEQ ID NOS 1-3 (accession No. L78223). Please see attached alignments.

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to combine the method of Hansen with the use of functionally equivalent primers selected from the sequence of Gonzalez and probe specific for this sequence since Hansen expressly teaches primer selection using Gonzalez's sequence with Genbank accession number L78223.

In the recent court decision *In Re Deuel* 34 USPQ 2d 1210 (Fed. Cir. 1995), the Court of Appeals for the Federal Circuit determined that the existence of a general method of identifying a specific DNA does not make the specific DNA obvious. Regarding structural or functional homologs, however, the Court stated,

"Normally, a *prima facie* case of obviousness is based upon structural similiarity, i.e., an established structural relationship between a prior art compound and the claimed compound. Structural relationships may provide the requisite motivation or suggestion to modify known compounds to obtain new compounds. For example, a prior art compound may suggest its homologs because homologs often have similar properties and therefore chemists of ordinary skill would ordinarily contemplate making them to try to obtain compounds with improved properties (see page 9, paragraph 4 of attached ref)."

Since the claimed primers and probe simply represent structural homologs, which are derived from sequences suggested by the prior art as useful for primers and probes for the detection of SUR1's "–3 nucleotide" of exon 16, and in particular for diagnosis of NIDDM and concerning which a biochemist of ordinary skill would attempt to obtain alternate compounds with improved properties, the claimed primers and probes are *prima facie* obvious over the cited references in the absence of secondary considerations.

Buck expressly provides evidence of the equivalence of primers. Specifically, Buck invited primer submissions from a number of labs (39) (page 532, column 3), with 69 different primers being submitted (see page 530, column 1). Buck also tested 95 primers spaced at 3 nucleotide intervals along the entire sequence at issue, thereby testing more than 1/3 of all possible 18 mer primers on the 300 base pair sequence (see page 530, column 1). When Buck tested each of the primers selected by the methods of the different labs, Buck found that EVERY SINGLE PRIMER worked (see page 533, column 1). Only one primer ever failed, No. 8, and that primer functioned when repeated. Further, EVERY SINGLE CONTROL PRIMER functioned as well (see page 533, column 1). Buck expressly states "The results of the empirical sequencing analysis were surprising in that nearly all of the primers yielded data of extremely high quality (page 535, column 2)." Therefore, Buck provides direct evidence that all primers would be expected to function, and in particular, all primers selected according to the ordinary criteria, however different, used by 39 different laboratories. It is particularly striking that all 95 control primers functioned, which represent 1/3 of all possible primers in the

target region. This clearly shows that every primer would have a reasonable expectation of success.

4. Claims 22-24 are rejected under 35 U.S.C. 103(a) as being unpatentable over Hansen et al(Diabetes, April 1998) in view of Gonzalez et al(Genbank accession no. L78223; SUR1 gene, exon 17, June 1996) and in further view of Buck et al (Biotechniques (1999) 27(3):528-536) and in further view of Gibco BRL catalog(Pgs. R-67, R-68, 1993-1994)and in an even further view and in further view of Ahern(The scientist, 1995).

With regard to claim 13, Hansen et al. teach that "a diminished acute insulin secretory response to intravenous glucose or sulfonylurea loads is a frequent feature of late-onset NIDDM"(Pg. 508). The reference further teaches this method comprising:

a) obtaining a sample from a NIDDM patient, said sample comprising nucleic acid molecules containing the fragment of the SUR1 gene comprising the nucleotide in position –3 of exon 16(Table 4, pg. 602),

b) detecting the presence or the absence of the –3t allele of exon 16, whereby the presence of at least one –3t allele identifies a NIDDM patient with a higher susceptibility toward sulfonylurea therapy(Entire document).

As a whole, the reference teaches that the "at risk genotype" for NIDDM is comprised by at least one –3t allele of exon 16, and that a characteristic of late onset NIDDM is a diminished response to sulfonylurea loads.

With regard to claim 14, Hansen et al. teach the above method further comprising prior to step b) the step of amplifying said nucleic acid molecules using amplification

primers that selectively anneal to and amplify a portion of said gene comprising the nucleotide in position –3 of exon 16(Table 1 and Research Design and Methods section).

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With regard to claims 19, 20, and 21 Hansen et al. teach the above method wherein said detecting step b) comprises obtaining a first gene fragment comprising nucleotide –3 of exon 16 isolated from said human sample and a second gene fragment comprising nucleotide –3 of exon 16 isolated from said human sample and a second gene fragment comprising nucleotide –3c and –3t of exon 16, said second fragment corresponding to said first fragment, forming single-stranded DNA from said SUR1 gene fragment and from said second SUR1 gene fragment, electrophoresing said single-stranded DNAs on said gel to determine if said single-stranded DNA from said first SUR1 gene fragment is shifted relative to said second SUR1 gene fragment, and optionally sequencing said single-stranded DNA from said first SUR1 gene fragment having a shift in mobility.(See Research Design and Methods and specifically Pg. 599 and results on Page 601)

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Gonzalez teaches the SUR1 gene's DNA sequence for SEQ ID NOS 1-3 (accession No. L78223). Please see attached alignments.

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In the recent court decision *In Re Deuel* 34 USPQ 2d 1210 (Fed. Cir. 1995), the Court of Appeals for the Federal Circuit determined that the existence of a general method of identifying a specific DNA does not make the specific DNA obvious. Regarding structural or functional homologs, however, the Court stated,

"Normally, a *prima facie* case of obviousness is based upon structural similiarity, i.e., an established structural relationship between a prior art compound and the claimed compound. Structural relationships may provide the requisite motivation or suggestion to modify known compounds to obtain new compounds. For example, a prior art compound may suggest its homologs because homologs often have similar properties and therefore chemists of ordinary skill would ordinarily contemplate making them to try to obtain compounds with improved properties (see page 9, paragraph 4 of attached ref)."

Since the claimed primers and probe simply represent structural homologs, which are derived from sequences suggested by the prior art as useful for primers and probes for

the detection of SUR1's "–3 nucleotide" of exon 16, and in particular for diagnosis of NIDDM and concerning which a biochemist of ordinary skill would attempt to obtain alternate compounds with improved properties, the claimed primers and probes are *prima facie* obvious over the cited references in the absence of secondary considerations.

Buck expressly provides evidence of the equivalence of primers. Specifically, Buck invited primer submissions from a number of labs (39) (page 532, column 3), with 69 different primers being submitted (see page 530, column 1). Buck also tested 95 primers spaced at 3 nucleotide intervals along the entire sequence at issue, thereby testing more than 1/3 of all possible 18 mer primers on the 300 base pair sequence (see page 530, column 1). When Buck tested each of the primers selected by the methods of the different labs, Buck found that EVERY SINGLE PRIMER worked (see page 533, column 1). Only one primer ever failed, No. 8, and that primer functioned when repeated. Further, EVERY SINGLE CONTROL PRIMER functioned as well (see page 533, column 1). Buck expressly states "The results of the empirical sequencing analysis were surprising in that nearly all of the primers yielded data of extremely high quality (page 535, column 2)." Therefore, Buck provides direct evidence that all primers would be expected to function, and in particular, all primers selected according to the ordinary criteria, however different, used by 39 different laboratories. It is particularly striking that all 95 control primers functioned, which represent 1/3 of all possible primers in the target region. This clearly shows that every primer would have a reasonable expectation of success.

Hansen in view of Gonzalez and in further view of Buck et al. do not teach a kit containing the above primers, a *Pst I* restriction enzyme, and instructions for the kit's use.

However, the Gibco BRL catalog teaches the recognition sequence of *Pst I* and as Hansen taught the use of a Restriction enzyme(Pg. 601 left side) to identify the SUR1 gene allele present, it would have been obvious in light of the sequence of Gonzalez to

select another restriction enzyme, namely *Pst I* that is specific to the Gonzalez's nucleotides present at the –3 location of exon 16 from the BRL catalog.

Hansen in view of Gonzalez in further view of Buck et al. and in an even further view of the Gibco BRL catalog, do not teach a kit comprising a pair of primers specific for amplifying all or part of the SUR1 gene comprising nucleotide –3 of exon 16 and instruction relating to detecting the presence of a –3t allele with a higher susceptibility toward sulfonylurea therapy.

Ahern et al. do teach that "more researchers are buying premade reagents and kits because they are convenient and they save time"(Pg. 4) The reference goes on to teach that "a kit that supplies all of the necessary reagents for a particular research application and even provides them with detailed instructions to follow"(Pg. 4).

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to combine the method of Hansen with the use of functionally equivalent primers selected from the SUR1 sequence of Gonzalez and probe specific for this sequence since Hansen expressly teaches primer selection using Gonzalez's sequence with Genbank accession number L78223 and further obvious to incorporate the primers, *Pst I* in view of the well known practice of choosing sequence specific restriction enzymes as taught in the Gibco BRL catalog(R-68), and instructions into a kit in further view of Ahern as she teaches a motivation for combining reagents into kits for the expected benefit that "the large selection of prepared biochemicals and kits for specific applications has certainly made life easier for countless researchers"(Pg. 5).

Applicant should note that with respect to their product claims:

A recitation of the intended use of the claimed invention must result in a structural difference between the claimed invention and the prior art in order to patentably distinguish the claimed invention from the prior art. If the prior art structure is capable of performing the intended use, then it meets the claim. In a claim drawn to a process of making, the intended use must result in a manipulative difference as compared to the prior art. In the instant case, using the "-3c" and "–3t" alleles of the SUR1 gene as the instructions direct in a kit is no different from the instantly claimed methods drawn to nucleic acid molecules of the SUR1 gene that are already taught in the prior art(see MPEP 2111.02).

Any inquiry concerning this communication or earlier communication from the examiner should be directed to Sally Sakelaris whose telephone number is (703) 306-0284. The examiner can normally be reached on Monday-Thursday from 7:30AM-5:00PM and Friday from 1:00PM-5:00PM.

If attempts to reach the examiner are unsuccessful, the primary examiner in charge of the prosecution of this case, Jeffrey Fredman, can be reached at (703)308-6568. If attempts to reach the examiners are unsuccessful, the examiner's supervisor, Gary Benzion, can be reached on (703)308-1119. The fax number for the Technology Center is (703)305-3014 or (703)305-4242.

Any inquiry of a general nature or relating to the status of this application should be directed to Chantae Dessau whose telephone number is (703)605-1237.

Sally Sakelaris

9/15/2003

JEFFREY FREDMAN
PRIMARY EXAMINER